REMARKS/ARGUMENTS

The Pending Claims

Claims 1, 3-26, 35-43, and 45-66 are pending. Claims 7, 17, and 47-62 are withdrawn as directed to non-elected subject matter. Claims 1, 3-6, 8-16, 18-26, 35-43, 45, 46, and 63-66 are currently under examination.

The Pending Claims

New dependent claims 55-66 have been added. New claims 55-62 mirror previously presented claims 45-54, but depend from claim 40 instead of claim 1. New claims 63-66 further characterize claims 1 or 40 by reciting additional aspects of the invention. The new claims are supported by the original claims and the specification, for example, at paragraph [0053] and the Examples.

Summary of the Office Action

The Office rejects claims 1, 4, 5, 8-10, 12, 13, 18-21, 23-26, 36, 38-41, 43, 45, and 46 under 35 U.S.C. § 103(a) as allegedly unpatentable over Wu et al., *Mol. Cell Biol., 22*: 7688-7700 (2002) in view of Bass, *Nature, 411*: 428-429 (2001) and U.S. Patent Application Publication 2004/0006005 (Bhanot) and further in view of Nicklin et al., *Curr. Gene Ther., 2*: 273-293 (2002), Sui et al., *Proc. Natl. Acad. Sci., USA, 99(8)*: 5515-5520 (2002), Parrish et al., *Mol. Cell, 6*: 1077-1087 (2002), U.S. Patent Application Publication 2004/0086884 (Beach et al.) and U.S. Patent 6,331,425 (Taylor et al.).

The Office objects to claims 6 and 16 as depending from a rejected base claim, but acknowledges that these claims would be allowable if rewritten in independent form.

Reconsideration of these rejections is hereby requested.

Discussion of the Obviousness Rejections

In setting forth the Section 103 rejections, the Office relies principally on Wu et al. for its disclosure of the MAML2 sequence, and the Bass and Bhanot references for disclosures relating to antisense or siRNA technology. In particular, the Office alleges that it would have been obvious, given the disclosure of these references, to use siRNA against

MAML2 in order to explore the function of the MAML2 gene. The Office further alleges, based on Bhanot, that one of ordinary skill in the art would have targeted the stop codon region of MAML2 with siRNA, thereby arriving at a nucleic acid molecule that inherently would comprise a portion of SEQ ID NO: 12 and inhibit a Mect1-MAML2 fusion protein. The other references cited by the Office are relied upon for the disclosure of elements recited in dependent claims. Applicants traverse the Section 103 rejection.

Without admitting that Wu et al. is prior art to the present application, the combined disclosures of the cited references would not have led one of ordinary skill in the art to the claimed subject matter at the time of filing. Assuming, for the sake of argument, that the prior art suggested using siRNA to test the function of MAML2,[1] not every siRNA designed to inhibit MAML2 would inhibit the Mect1-MAML2 fusion gene. Mect1-MAML2 comprises only part of MAML2. Thus, if one of ordinary skill in the art applied siRNA to MAML2 at the relevant time, they would not necessarily have arrived at a nucleic acid that would inhibit Mect1-MAML2.

In order to address this deficiency in the prior art disclosures, the Office argues that Bhanot more specifically discloses targeting the stop codon region of an mRNA transcript with siRNA, and that by targeting the stop codon of MAML2 one of ordinary skill in the art would arrive at a molecule that inherently would inhibit the Mect1-MAML2 fusion gene.[2] Applicants respectfully disagree with the Office's argument for several reasons.

Contrary to the Office's argument, Bhanot does not suggest targeting the stop codon region over any other portion of an mRNA transcript. The cited passage of Bhanot, when read as a whole, states that *any* portion of the mRNA molecule considered in that reference may be targeted:

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[1] We disagree with this contention. Neither Wu et al. nor any other reference cited by the Office discloses siRNA molecules directed to MAML2. Rather, the Office assumes that siRNA would be an appropriate technique for testing the gene function of MAML2 based on generic disclosures that siRNA is useful in other contexts (e.g., Bass et al. and Bhanot et al.). At the relevant time, a vast number of methods were available for such purposes, several of which were employed by Wu et al. Nothing in Wu et al. or any other reference cited by the Office suggests that siRNA would be more appropriate than such other methods.

[2] The Office apparently assumes, without citing any support, that Mect1-MAML2 contains the stop codon region of MAML2.

> For example, an antisense compound of the present invention comprises about 8 to about 80 linked nucleobases targeted to nucleobases of *a start codon, a 5' UTR region, a coding region, a 3'UTR region, or a stop codon* of a nucleic acid molecule encoding human Integrin-linked Kinase ... . (emphasis added)

(Bhanot at para. [0012]). The regions mentioned in the above passage constitute the entire mRNA transcript.

Furthermore, nothing in Bhanot suggests targeting the stop codon region over any other region of an mRNA transcript. In fact, Example 15 of Bhanot shows that, for the gene of interest to Bhanot, stop codon-targeted siRNA's are no more preferable than siRNA's directed to the start codon or coding sequences (Bhanot at paras. [0286]-[0290] (Example 15 and accompanying Table 1)).

The Office apparently selected the "stop codon" disclosure of Bhanot, ignoring the disclosure as a whole, in order to conform to the teachings of the present application. Specifically, Applicants disclose that Mect1-MAML2 comprises exons 2-5 of MAML2, i.e., the "tail end" of the mRNA transcript. The Office evidently presumes, based on this disclosure, that Mect1-MAML2 contains the stop codon region of MAML2. Using this information, the Office has isolated a portion of a prior art dislcosure to support the rejection.

However, the present patent application is not prior art to the claimed invention. One of ordinary skill in the art at the relevant time would not have had the benefit of knowing that exons 2-5 of MAML2 were part of the Mect1-MAML2 fusion gene product. Without such information, one of ordinary skill in the art applying siRNA to MAML2 would not necessarily be led to the claimed subject matter.

Moreover, the disclosure of Bhanot pertains only to the Integrin-linked Kinase gene. Any disclosure as to which regions of Integrin-linked Kinase are effective siRNA targets is simply not applicable to a completely different gene, such as Mect1-MAML2.

When properly considered, the cited references do not suggest the claimed subject matter. For this additional reason, the Section 103 rejection of the claims should be withdrawn.

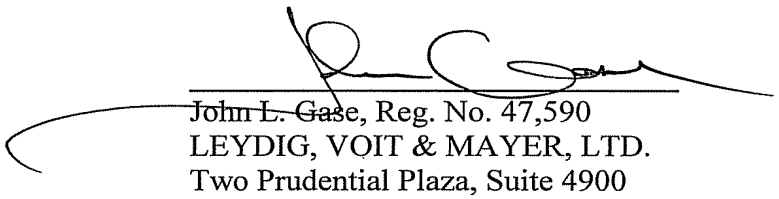
Discussion of the New Composition Claims

Claims 63-66 further characterize the compositions of independent claims 1 and 40, respectively, by stating that the composition inhibits the growth of a cancer cell comprising a Mect1-MAML2 chimeric gene. Applicants believe the subject matter of these claims is even further removed from the prior art.

Conclusion

Applicants respectfully submit that the patent application is in condition for allowance. If, in the opinion of the Examiner, a telephone conference would expedite the prosecution of the subject application, the Examiner is invited to call the undersigned attorney.

Respectfully submitted,



John L. Gase, Reg. No. 47,590
LEYDIG, VOIT & MAYER, LTD.
Two Prudential Plaza, Suite 4900
180 North Stetson Avenue
Chicago, Illinois 60601-6731
(312) 616-5600 (telephone)
(312) 616-5700 (facsimile)

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